January 10, 2012, Updated March 8th, 2012

RE: Jennifer Peterson, DEQ, DRAFT Comments on Appendix G, Baseline Ecological Risk Assessment, July 1, 2011, Prepared for the Lower Willamette Group by Windward Environmental.

General Comments:

1. Dioxin TEQ: The document confounds sources of total dioxin TEQ by not properly discussing the sources that contribute - dioxins and furans (dioxin TEQ) and dioxin like PCBs (PCB TEQ). It is true that PCBs are a major driver, but this is not the case near RM 7 near the ARKEMA facility this source is driven by dioxin TEQ. This should not be lost by focusing solely on PCBs.

2. Spatial Scale and Proper Alignment of Lines of Evidence: The document is focused on developing a list of COPCs for each receptor group and media. The COPC identification process focuses on data rules that extend beyond an evaluation of those receptor / contaminant pathways that have a hazard quotient greater than one. ALL combinations with an HQ>1 should be clearly outlined and more importantly described between different media and receptors such that ecological lines of evidence are clearly articulated. While the description of each receptor group is necessary, the more important high level analysis is the integration of lines of evidence between different media and receptor groups on an appropriate spatial scale.

3. Identification of COPCs: The lack of inclusion of surrogates for components of some risk drivers is likely confounding and underestimating risk, particularity for butyltins and dioxins and furans other than 2,3,7,8-TCDD. This is also confounding the comparison between different receptors and media lines of evidence since in some cases these important COI were not further investigated or included in the COPC summary tables.

Specific Comments:

Table 4-2, RM 11 E Sediment, Footnote "h": Sediment from this river mile were only used "for the benthic community evaluation in order to be consistent with the data lockdown agreements between the LWG and EPA. " Omission of these data from other lines of evidence in the ERA could underestimate risk to the sources in this area.

Table 4-3, Round 3B Biota Sampling, Smallmouth Bass: The range given is from 225 to 355 mm. This is not the correct range retained in the Round 3 FSP and in fact larger fish were not included on a site wide basis although they were caught in order to be consistent with the smaller range fish that were collected during Round 1. Fish larger than 335 mm were not routinely retained and included in composite samples.

Section 4.1.3, Bird Egg Tissue, Footnote 33, Diphenyl Ethers: It is unclear why these data were not included in the SCRA and assigned a QA/QC level of Category 2. This designation should be reviewed given the source of polybrominated diphenyl ethers at the site.

Section 4.2, Non-Study Area Data: It is unclear why the downstream reach includes RM 1.9 which is co-located with Oregon Steel Mills. "Non-study" area data should be presented in the main text of the BERA and not delegated to Attachment 4.

Section 6.5.5.2.2, Uncertainty Analysis of Surface Water Sampling Methods: All surface water samples regardless of the sampling protocol (XAD or peristaltic pump) should be included in the screening and used to determine COPCs (samples with HQ>1). Instead, some peristaltic samples were only included in an evaluation of uncertainty. The fact that XAD has lower detection limits does not invalidate the results of peristaltic samples that captured exceedances of water quality thresholds at different sampling locations, positions in the water column or time of year. Some samples that have been excluded:

> 4,4'-DDT and Total DDX: W027 (Mult. Channel) and W031 (GASCO) were exceeded by a greater magnitude (by one order of magnitude) than XAD samplings in that location during different sampling periods.
>
> 4,4'-DDT: Two additional peristaltic sample locations – W030 at RM 5.5 and W036 at RM 8.6 exceeded benchmarks and should be included in the list of locations where HQ's >1.

Section 5.1.2, Refined Screen: It is still unclear if the decision criteria outlined in the flowchart were appropriate. As a part of resolutions on comments from the XXXX DRAFT BERA, a table showing each chemical screened out of the refined screen and the reason was supposed to be developed. This information was not included in the revised document. The flowchart indicates that chemicals were screened out based on frequency of detection, which may not appropriately consider appropriate spatial scale.

1. Benthic Invertebrates, Section 5.2: Samples here the detection limit exceeded the screening level TRV should be retained in the SLERA. Dropping COIs at this stage does not allow for proper alignment of different lines of evidence in the risk assessment. In addition, COIs without SLVs should be retained in the SLERA and analyzed with other lines of evidence where SLVs are available. COIs dropped include:
    a. Sediment (occurred 30% of the time): Diethyl phthalate, dimethyl phathalte, 1,3-dichlrobenzene, and heptachlor
    b. Crayfish (occurred 80% of the time): dibutylphalate and dimethyl phthalate

2. Fish (See Table 5-5):
    a. Dietary: Monobutyltin, dibutyltin, and tetrabutyltin should be included in these counts separately even if tributyltin is used as a surrogate for effects. The assessment of tributyltin specific concentrations should not be assumed to cover those of the other butyltins.
    b. Tissue (17-57%): butyl benzyl phthalate, dibutylphthalate, diethylphthalate, hexachlrobutadiene, endrin, alpha-HCH, beta-HCH and delta HCH
    c. Surface Water (30%): 2,4-DDE
    d. TZW: Selenium and styrene

Table 5-3, Benthic Invertebrate COIs with No TRVs: Every effort should be made to include relevant SLVs in the screening stage. For example, it is unclear why there is not a SLV for tributyltin in sediment when several exist, including in DEQ guidance (use marine AET as a surrogate). Tributyltin should be used as a surrogate for the other butyltins in screening. DEQ also has a sediment screening value for dioxins (based on a TEQ) from the NOAA Squirts table. EPA Region 3 also has a comprehensive list of sediment benchmarks that would fill many of these gaps available at: http://www.epa.gov/reg3hwmd/risk/eco/btag/sbv/fwsed/R3_BTAG_FW_Sediment_Benchmarks_8-06.pdf

Table 5-4, Fish COPCs: Why is aluminum omitted from the list of COPCs from surface water and TZW?

Table 5-6, Fish COIs with no TRVs:

1. Tissue: TRVs for tissue for tributyltin should be used as a surrogate for monobutyltin ion and dibutyltin ion for assessing fish tissue concentrations similar to Table 6-3 for surface water. These are important as they are detected at significant concentrations in fish tissue (e.g. TRV excedance in fish for monobutyltin, Table 7-37) which is co-located with the surface water exceedances.

2. Transition Zone Water: TRVs for TPH in TZW: TRVs are available for TPH in transition zone water and were provided by EPA.

3. Surface Water: TRVs for surface water for 2,4-DB and MCPP are available:
   a. 932 ug/L, LOAEL for green algae (*Selenastrum capricornutum*) from the Environmental Fate and Effects Division's Risk Assessment of the Reregistration Eligibility Document for 2,4-DB (EPA 2005). This comment also applies to Table 5-12.
   b. MCPP: 2.60 ug/L using MCPA as a surrogate, a Canadian Water Quality Guidance Surface Water Quality Screening Level Benchmark. This comment also applies to Table 5-12.

Table 5-6, Wildlife COPCs: Dibutylphalate should be identified as a COPC for osprey given the detection limit was above the screening level. Justification is provided that inclusion is unnecessary based on the fact that sediment threshold concentrations were not exceeded regardless of other lines of evidence that identify elevations and exceedences of this contaminant in other prey tissue. Also, 40% of the non-detected carp tissue had detection limits >than the oprey tissue threshold making the elimination of this COPC in the refined screen a highly uncertain determination. It is also not clear why this contaminant was not carried over from the SLERA for other wildlife receptors (mink and river otter) – see footnote a.

Table 5-9, Wildlife COIs With No TRVs:

1. Silver: Wildlife TRVs for mammals and birds are available from EPA's Eco SSLs at: http://www.epa.gov/ecotox/ecossl/pdf/eco-ssl_silver.pdf
2. Dibenzofuran: Mammalian TRVs are available as a part of EPA's PPTRVs of 0.123 mg/kg/d

Section 6, Benthic Invertebrate Assessment: Comments on this section are not completed due to the review of the predictive benthic models.

Section 6, Objectives of FPM Model Selection and Risk: This section states "the FPM with the most balanced error rates and the LRM selected by EPA were carried forward to help assess benthic risk". Since the FPM with the most balance error rates is not model selected for the risk assessment but rather

for the feasibility, this section will need to be carefully worded to ensure the reader does not assume that SQVs developed from a model with balanced rates ***is also indicating appropriate risk thresholds / areas***.

Section 6, Use of Mean Quotients Using Site-Specific SQVs: The use of mean quotients is a way to combine ***contaminant specific independent SQGs*** into a mixture model specific to the range of contaminants at a given site. ***This methodology should not be used to combine a set of SQVs that are not independent***, such as those that come out of the floating point model. For the FPM, the exceedance of any SQV in the set would indicate toxicity. This analysis should be limited to the national freshwater SQGs as was originally directed by EPA. EPA's direction was to use a mean quotient threshold of 0.7 to evaluate national SQGs – it should not be further interpreted that a mean quotient of 0.7 is appropriate for use with dependent site specific SQVs from Portland Harbor.

Section 6.1.1.3, Uncertainty Analysis of Invertebrate Sediment Toxicity Assessment: Comment Placeholder.

Section 6.1.1.4, Summary of Invertebrate Sediment Toxicity Assessment: Comment Placeholder.

Section 6.2, Predictive Benthic Toxicity Models: Comment Placeholder.

Section 6.2.1-6.3 and Section 6.7: Comment Placeholder – see draft comments / questions.

Section 6.4.1, Tissue-Residue Risk Assessment Methods: A TRV exceedance in one sample should be interpreted as posing an unacceptable risk to the benthic community (or bivalves or crayfish) in a given area. Invertebrates are immobile or nearly immobile and therefore the proper exposure point concentration is a point by point evaluation. Furthermore, effects on benthic invertebrates have cascading effects on nutrient cycling and fish resources that extend beyond risk to invertebrate populations themselves. The protection of a benthic community transcends assessment endpoints to effects on amphibians, fish and wildlife.

Section 6-12, Benthic Invertebrate Tissue COIs with No TRVs, Dioxins / Furans: Sediment SQGs are available for dioxin TEQ (NOAA Squirts, Canadian Sediment SQGs and DEQ guidance) and should be used. Alternatively, the 2,3,7,8-TCDD value can be used as a surrogate for the other dioxin and furan congeners.

Section 6.4.5.4, Butyltins: This section concludes that the risk of the other butyltins are covered by the evaluation of tributyltin (e.g. monobutyltin ion, dibutyltin ion and tetrabutyltin ion). However, the concentrations of the other butyltins are significantly higher and are shown to be correlated with benthic toxicity and elevations in aquatic tissue. Based on this, the aquatic toxicity framework used in the BERA should be re-evaluated to ensure the toxicity of all butyltins are properly evaluated.

Section 6.5.4, Effects Assessment, Butyltins: Monobutlyin is showing up in surface water and also appears to be a driver in sediment toxicity (see draft comments on predictive model) and detections in tissue. For example, this butyltin was detected in clam tissue at 97 ppb; dibutyltin at 560 ppb. Lines of evidence need to be integrated.

Section 6.5.4, Effects Assessment, Fish, DDx and PCBs: Have the alternative water quality criteria calculated by LWG been reviewed and accepted by EPA? These alternative values are the only values used to "determine risk conclusions to fish".

Section 6.0, Tables 6.4 and Text: Samples from the peristaltic pump water samples excluded from the risk assessment (Section 6.5.5.2.2) should be added into these tables and list of samples with HQ>1. These samples have not basis being relegated to the uncertainty section, as there is no additional uncertainty in the data quality or applicability of these samples.

Section 6.5.5.4, COIs for Which Cannot Be Quantified: See previous comments on TRVS and use of surrogates.

Section 6.6.2, Table 6-37:

a. Why were the 2,4' and 4,4' isomers of DDD, DDE and DDT (except 4,4,'-DDT) removed from the table?
b. Aluminum is removed from the COPC table based on a withdrawl of the AWQC because it was developed "using toxicity information from acidic waters and is not applicable to the circumneutral waters of the study area". Is this accurate? Some areas do meet these criteria, such as samples off ARKEMA where the pH is very acidic. These are also areas where exceedances occur (e.g. off ARKEMA's chlorate plant). Exceedances appear to occur co-located with contamination that likely alters the chemistry and pH in the area (e.g. bulk fuel facilities, GASCO).
c. Dioxins and furans: Dioxins in addition to 2,3,7,8-TCDD should be carried forward using the TCDD AWQC as a surrogate. This is appropriate given the site risk is driven by the significant elevations of dioxins and furans other than 2,3,7,8-TCDD and results in these additional dioxin / furans included in the Summary tables (Table 6-39).
d. SLVs for the complete list of TPH should be added to this table – only gasoline-range is currently included. In addition, gasoline range TPH should be included in the final count of COPC exceedances. Footnote a indicates it was excluded.

Section 6.6.3.3, Uncertainty Associated with Ecological Exposure to TZW: This section is biased toward the potential exposure of one group of invertebrates without considering that many species of insects and invertebrates (and ammocoetes) do not utilize tubes or burrows. Furthermore, there is not sufficient evidence that those that do use burrows are not also exposed to contaminated surface and groundwater surrounding them.

Section 6.6.3.3, Table 6-40: Please note that this table only considers the alternative water TRVs for DDx (0.011 ug/L). It is also unclear why individual isomers other than 4,4'DDT were removed from the analysis. Each isomer and Total DDX water value should be compared to the water quality criteria.

Section 6.6.5.1, Table 6-41: Individual Isomers of DDT should added back into the table (2,4'DDD, 2,4'DDT, 4,4'-DDD, 4,4'-DDE) as indicated in Figure 6-25.

Section 6.6.5.3, Table 6-42: TRVs for total petroleum hydrocarbons (TPH, residual, diesel range, etc.) should be added to the table. There is a water threshold available. 2,3,7,8-TCDD water value should be used as a surrogate for the other congeners.

Section 6.6.2, Table 6-3, COIs without Screening Level Benchmarks: Benchmarks are available for aluminum, and residual range, diesel range, diesel residual range and total PAHs.

Section 7, Fish Risk Assessment:

Spatial Scale and Determination of COPCs – Tissue Residue Line of Evidence(Section 7.0) – see also the removed Section 2.0 by examining the red-lined version from Attachment 12: There appear to be new steps introduced into the risk assessment that expands the spatial scale for the evaluation of the fish tissue residue line of evidence. Further averaging is conducted beyond a composite by composite analysis (step 1) that was presented in the previous DRAFT BERA. Table 7-7 should be used to determine COPCs (Step1) and not Table 7-1 (Step 2) which expands the spatial scale. Composite samples already represent an average concentration over a relevant spatial scale that was selected according to the home range of the fish. COPCs should be identified based on Step 1 (Sample by sample basis) instead of further widening the scale beyond the home range of the fish in Step 2 (Table 1). They are citing an agreement made on Oct. 15th 2010 as justification? See also Section 7.1.5 (risk characterization) and in particular the text that was removed. These include:

Sculpin: Previously was composite by composite. Samples removed:
- Copper: 3 composite samples with HQs>1 (out of 38)
- PCBs: 4 composite samples with HQs>1 (out of 38)
- 4,4'-DDT: 1 composite samples with HQ>1 ((out of 38)
- Total DDX: 1 composite samples with HQ>1 ((out of 38)
- BEHP: 1 composite sample with HQ>1 ((out of 38)

Lamprey: Site-wide exposure, previously composite by composite. Samples removed:
- Copper: 4 composite samples (out of 4)

Largescale Sucker: Previously 3 mile composite by composite; now site wide. Samples removed:
- PCBs: 2 composite samples with HQs>1 (out of 6)

Peamouth: Previously 3 mile composite by composite; now site wide. Samples removed:
- Lead: 1 composite with HQ>1 (out of 4)

Smallmouth Bass: Previously composite by composite; now averaged with other composites in arbitrary 1 mile (both side of the river) scenario. Composites should not be averaged with other composites as they already represent the appropriate spatial scale. Samples removed:
- Lead: 2 composites with HQ>1 (out of 32)
- PCBs: 9 composite samples with HQs>1 (out of 32)
- BEHP: 2 composite samples with HQs>1 (out of 32)

Northern Pikeminnow: Previously composite by composite as fish were composited over 3 miles; now averaging over the site.

Mercury: 1 composite with a HQ>1 (out of 6)

Total PCBs: 2 composites with a HQ>1 (out of 6)

Fish, Dietary Dose Line of Evidence: Sample by sample HQs in Appendix 12, show relevant spatial scale to different fish receptors of concern. However, these were then carried forward to be analyzed in a spatial scale larger than the home range of the organism by looking at a site-wide exposure scenario for all fish. What happened to TBT? Why are all the TBT HQs for fish changed to mercury? Also, the arbritary river mile breakpoints have a high potential to bifurcate known sources and average out exposure to fish prey and sediment. A sensitivity analysis should be performed on the analysis to determine how the results would change if different designations were made (esp. Willamette Cove area for mercury). Those removed by widening the spatial scale for the fish evaluation:

Smallmouth Bass:

TBT, Swan Island Lagoon: Lab worm HQ of 45 for prey for fish; sediment HQ of 1.5

Cadmium: RM 1.5 to 2.5; RM 3.5 to 4.5

Copper: All river miles

Others:

Contrary to the previous draft where COPCS were "identified as those COPCs with HQs >1 based on ecologically relevant exposure scales for sculpin, smallmouth bass, and northern pikeminnow because fish samples were collected and composited over areas that represent what are conservatively assumed to be ecologically relevant exposure scales" (previous version). There now appears to be no analysis of composite by composite screening since COPCs are based on Step 2 (averaging over larger spatial scales).

Section 7.1.2, COPCs Evaluated: Monobutyltin (and other butyltins like dibutyltin) should be evaluated given their detections in fish tissue. Use a surrogate, but it is not appropriate to relegate them to "COIs without screening level TRVs". For example, monobutyltin was detected in juvenile salmon at a max concentration of 5.6 ug/kg.

Section 7.1.3.1, Empirical Tissue EPCs: There is a citation provided that under EPA guidance both composite and discrete samples are appropriate for calculation of UCLs using Pro UCL Software (EPA 2007b). This guidance is directed at calculating soil exposure point concentrations and not body burdens for the protection of fish populations. The calculation of exposure point concentrations for the protection of fish should be on a relevant spatial scale which is composite by composite, as the composites were already designed with home range in mind.

Section 7.1.3.2, Predicted Tissue EPCs: Predictions of dioxins and furan concentrations for sculpin should be completed. If the mechanistic model is not used (footnote 9), then there are other models that can be used such as biota sediment accumulation factors. It appears that no bioaccumulation model was developed for dioxins and furans. Additionally, there is a danger of not further assessing COPCs for which no BSAR could be developed, such as BEHP. A site wide BSAR may not be appropriate given the range and unique chemical signatures across the sites. However, localized areas within the site can be evaluated using a BSAF.

Table 7-5, Selected Whole-Body Tissue TRVs:

Mercury: This TRV is increased by eliminating adverse effects associated with egg or embryo residues. This is not appropriate and was not a EPA comment on the previous draft. The LWG is implying that exposure to these life stages is not "directly comparable to the contaminant concentration data for the fully formed fish that were used to characterize receptor exposure in the Study Area". The fact that all sensitive life stages were not collected and analyzed should not preclude the assessment of these stages in the risk assessment. This change is not a result of an EPA comment on the previous draft so it is unclear why this change was made.

Section 7.1.3.2, Effects: Contrary to the text in this section, effects from exposure to multiple chemicals that share the same mode of action should have been factored into the risk assessment. This would be particularly important in the assessment of dioxin like toxicity attributed to the combined effect of dioxins, furans and PCB congeners.

Section 7.1.4., Fish Whole –Body Tissue Residues and TRVs:

a. Buyltins should have been carried forward from the SLERA and tissue residue TRVs developed for fish.
b. Removal of Embryo and Egg Toxicity Information: Several fish TRVs have been changed based on the removal of egg or embryo residue data. Although the residue we currently have for the site is whole-body fish, these residues are an indication of levels that could also accumulate in embryos and eggs and these evaluations are also meant to be protective of amphibians. These life stages may accumulate these contaminants to greater concentrations given they are in direct contact with the sediment and do not average exposure over larger areas. Since the protection of eggs and embryos is an assessment endpoint, these values should be included in the development of tissue residues for the protection of fish.

Section 7.1.5.1, Antimony: Elevated risk is attributed to potential fish sinker. However, there are sediment samples that are elevated for antimony in the area where the composite was taken (RM 9.5 to 10.5).

Section 7.1.5.1, Mercury: Mercury should not be dropped as a COPCS for fish based on the change in the TRV. The area where the elevation occurs (RM 6.5 to 7.5) is an area of elevated mercury in sediment and riverbank soils (Willamette Cove). This should be retained to line up with other lines of evidence in area (e.g. risk to fish eating birds).

Section 7.1.5.1, PCB SSD: It appears the uncertainty in the statistical model selected could result in a decrease in the TRV to 0.76 mg/kg. The 0.76 mg/kg should be used in the risk assessment.

Section 7.1.5.2.1: The presentation of downstream and downtown reach (RM 11.8-15.8) that are so close to the study area in an Attachment (4) is confusion and does not allow the reader to interpret the distribution of risk using these data. It also appears that these data were not screened through the SLERA and Refined Screen, but rather COPCs identified for the study area were reported for the upstream and downtown reaches. It is unclear if additional COPCs would have been identified for these areas.

Section 7.1.5.2 and Table 7-11: The text states "this information can be used to compare the Study Area to upriver locations". Based on the size of fish collected in the upriver location compared to the Study

Area these comparisons are not possible. The size of the fish are significantly greater in the upriver locations and comparable sizes from the Study Area are not available (esp. for bass).

Section 7.1.5.2 and Table 7-12: it is unclear why this table comparing BEHP between the Study Area and the Upriver tissue was added to the BERA as it was not requested by any of the previous comments. Again, there are problems with establishing the upriver dataset as appropriate for comparisons to Study Area fish tissue concentrations. BEHP may be a COPC that does not show distinct differences in concentrations between older, larger fish and smaller, younger fish but for other more bioaccumulative chemicals such as PCBs, organochlorine pesticides, dioxins and furans and mercury.

Table 7-13, Fish Tissue COIs: It is unclear why PAHs are not on this list, as they were detected in Round 3 fish tissue. If they were screened out, it is also unclear what TRVs were used to do so. There are also chemicals on this list where preliminary risk evaluation TRVs were provided (e.g. AWQC x BCF methodology) such as Endrin. It is unclear if these chemicals were evaluated for a baseline TRV development using species sensitivity distributions.

Section 7.1.5.5, Evaluation of Non-Target Ecological Receptors: The spatial extent (location of composites) for BEHP and PCBs identified as COPCS for brown bullhead are not outlined, so it is not possible to determine if the selected fish receptors are protective of bullhead as the text indicates.

Section 7.2.1, Fish Dietary Risk Assessment: Dietary risk conclusions should be based on Step 2 – derivation of HQs over a relevant exposure area for individual prey and species as outlined by EPA's Problem Formulation. The only justification for basing the conclusions on Step 3 (the derivation of HQ's over a relevant exposure area accounting for the ingestion of multiple prey species). Additionally, it is unclear from this document what "multiple prey species" were used. The only justification for this change is footnote 28 "as agreed to between EPA and LWG on October 15, 2010".

Section 7.2,1, Attachment 5, Table 4-3: The footnote reads: "Note: The following chemicals were not identified as fish dietary COIs because while these chemicals were detected in sediment, they were not analyzed in tissue: barium, beryllium, calcium, hexavalent chromium, cobalt, iron, magnesium, potassium, sodium, tin, titanium, vanadium, 1,6,7-trimethylnaphthalene, 1-methylphenanthrene, and 2,6-dimethylnaphthalene." Even if there is not estimate of tissue concentrations, the sediment threshold concentrations can be developed and used to screen sediment for fish effects.

Section 7.2.2, COPCs Evaluated: The omission of mono, di, and tetrabutyltin from the evaluation because "no LOAEL was available from the literature" leaves a significant data gap. A **NOAEL** should be used if no LOAEL is available, and would be relevant to use for the protection of threatened and endangered species such as juvenile salmonids. Concentrations of mon, di and tetrabutyltin are expected to be significantly higher than TBT, so it is unclear if ONLY evaluating TBT "covers" risk from the other butyltins. Attachment 5, Table 4-4 shows all the butyltins with the screening value of 0.03 µg/kg bw/day using TBT as a surrogate. It is unclear why this was not carried through the risk assessment and the uncertainty discussed. This is converted to a Tissue Threshold Concentration (TTC) of 594 ug/kg wet weight (Table 4-6 using Largescale sucker as an example; using other fish the TTC is even lower). Since the maximum monobutyltin concentration in fish tissue was **3,600 ug/kg** (well above the TTC) this deserves further discussion in the risk assessment.

Section 7.2.3.2.1, Exposure Parameters: Risk estimates should be based on a reasonable temperature, as indicated in previous comments. Temperature significantly impacts fish ingestion rates, and the

temperature used here of 13.4 C underestimates temperatures during a significant portion of the year which are 16.2 C (EPA recommended). ***Section 7.2.5.4.3 outlines the uncertainty between using the two different water temperatures*** and finds the hazard quotients would increase by 16% to 17%. The new hazard quotients should be used. While the same COPCs may be identified as the text indicates, the areas of concern would likely increase – also an important component of the risk assessment to clearly identify.

Section 7.2.3.2.1, Exposure Parameters: % Moisture in prey is only indicated for tissue and not for sediment. Sediment specific % moisture should be used since prey and sediment are separated in the dietary dose equations.

Section 7.2.3.2, Table 7-18, Receptor Specific Prey Species: It is unclear why northern pikeminnow is assumed to eat sucker, carp, peamouth and other pikeminnow. The fish dietary assessment applies to contaminants that are metabolized (e.g. PAHs) or regulated (e.g. metals). Therefore, the inclusion of larger fish in the dietary estimates where accumulation of these COIs in tissue is not expected is not relevant to the assessment. In addition, the assumptions of what fish prey the fish receptors of interest are consuming is not defensible. This should be removed and the fish dietary assessment based on the consumption of Step 2 as outlined by EPA which incorporates primarily invertebrate prey should be used.

Section 7.2.5.1, Large Home Range Fish and Section 7.2.5.1.2 Small homeranged fish, Footnotes 34, 36, 37, 39, 40, 42, and 43: The footnotes state "monobutyltin, dibutyltin and tetrabutyltin were not included in this count because TBT was used as a surrogate". This is not accurate***. TBT toxicity was not used as a surrogate to evaluate the potential effects associated with concentrations of the other butyltins as the footnotes imply.*** Instead, these buyltins were dropped from the risk assessment and it was assumed that by only assessing TBT the others were addressed. Since there are significantly higher concentrations of the other butyltins present in tissue and the environment, the concentrations of these butyltins should be evaluated as independent COIs. Please see attachment 5 for some of the tissue concentrations. It should also be noted that all butyltins screened through the SLERA and refined screens for dietary risk to fish.

> ****Please note that monobutyltin was also identified as a surface water COPC for fish. Multiple lines of evidence would indicate this should be investigated further for the dietary assessment.

Section 7.3.1, Fish Surface Water Risk Assessement Methods, Step 2: Step 2 is added as "agreed to between EPA and LWG on October 15, 2010" (footnote 47) in which surface water data are averaged over larger areas of the river and not evaluated on a location by location basis. This additional step does not make sense, given the small home ranges of different fish (e.g. sculpin, bass) and the wider spatial resolution of the collected surface water samples. ***Potentially unacceptable risk should be identified based on those COPCS that resulted in HQ>1 in Step 1, according to prior direction and the problem formulation.*** Averaging water samples does not appropriately correspond to protection of small home range fish receptors. Water samples in the new LWG Step 2 were averaged according to Table 7-1 (see also comments on Section 7.0 on appropriate fish exposure scale). These areas include averaging surface water over 1 mile (both sides of river) and site wide exposure areas. This is without consideration of ***acute water quality criteria***, which could be exceeded on localized basis and should be evaluated.

Section 7.3.3.1, Surface Water EPCs: Peristaltic Pump Samples should be included in risk estimates. The reason for removal is that the XD samples achieved lower detection limits. This fact does not discount the results of peristaltic samples taken at different times, in different locations, or different sampling depths than the XAD. It is also not a commonly held position that the peristaltic samples are less representative of exposure than spatially and temporally averaged concentrations when assessing effects to aquatic life.

Section 7.3.5.1 and Table 7-36, Number of Individual Surface Water Samples with HQs>1: This table should also provide locations where the exceedences occurred.

Section 7.3.5.1 , Table 7-37, Summary of Site Wide Surface Water UCL HQs: This table should not replace Table 7-36 by discounting the localized area exceedences, but instead should highlight that DDX is a ***site wide risk driver for fish effects***. Again, both XAD and peristaltic samples should be used. For DDx, this will identify additional areas than the XAD analysis (e.g. Mult channel area).

Section 7.3.5.1.2, Monobutyltin: The text on the potential overestimation of risk of monobutytin does not indicate that exceedences of the TRV could indicate exposure (and conversion) from an initial exposure to TBT. Therefore, the use of a TBT surrogate may actually be a more accurate approach.

Section 7.4.5.1, TZW Risk Characterization Results, Table 7-42, Footnote f: The footnote states "because petroleum compounds are not CERCLA contaminants, gasoline-range hydrocarbons are not included in the final count of contaminants posing potentially unacceptable risk; they are included because they may nonetheless contribute to risk". This is not an accurate statement and should be removed here and the additional places in the document (e.g. subsequent tables).

Section 7.6.3, Table 7-46, Summary of Fish COPCs>1: The table should be re-done to indicate COPCs based on the appropriate spatial scale including localized screening composite by composite for fish, spatially distinct surface water samples (location by location), and using Step 2 of the fish dietary assessment as directed by EPA's problem formulation.

Section 7.6.3, Table 7-46, Summary of Fish COPCs>1: Footnote "J": The footnote indicates that "monobutyltin could not be evaluated because not LOAEL TRV was available from the literature. A LOAEL TRV was available for TBT. Because TBT is the most toxic butyltin, risks from monobutyltin is assumed to be lower than those of TBT. TBT screens out in Step 1". This does not consider the significantly higher concentrations of monobutyltin in fish tissue, which is not covered by only looking at TBT. Include a NOAEL for monobutyltin or use the TBT TRV as a surrogate for concentrations detected in fish tissue. Also, it should not noted that multiple lines of evidence point to monobutyltin as a COPC (fish dietary – if it was evaluated, fish surface water, fish tissue).

Section 7.6.3, Table 7-46, Summary of Fish COPCs>1: Footnote "f", PAHs: "PAHs were not a COI for the tissue residue LOE because fish metabolize PAHs". PAHs were detected in fish tissue in Round 3 and concentrations should have been discussed in the risk assessment. The detection of PAHs in fish tissue indicates that exposure and metabolism to more toxic breakdown products is occurring. The concentrations detected in tissue are likely an underestimate of risk and should be discussed. This information should also be used in conjunction with other lines of evidence such as surface water.

Section 7.6.3, Table 7-46, Summary of Fish COPCs>1: Footnotes "d and g", Alternative Water TRVs for PCBs and 4,4,'DDT: Only the alternative PCB TRV HQ's are presented in this table. HQs based on the AWQC should be presented.

Section 8, Table 8-3, Shorebird Exposure Areas: The problem formulation calls for shorebird exposure areas in 1-mile increments. However, the exposure area has been changed in the ERA to 2 mile exposure areas calculated using 90% UCLs. Since many beaches are contaminant specific, this methodology in some cases bifurcates or dilutes out ecologically relevant concentrations.

Section 8.0, Wildlife Risk Assessment: The text states "risk conclusions were based on the final step (i.e. step 3 for the dietary LOE)" "***as agreed to between EPA and LWG on October 15, 2010 meeting*** (footnote 5 and Section 8.1.1 footnote 6)". Risk concusions should be based on step 3 unless the range of receptor prey species is varied probabilistically as indicated by the problem formulation. There is too much uncertainty in attaching simple prey portions especially for receptors like the hooded merganser. The proportions presented imply an inaccurate precision. Risk characterization should be based on Table 8-15, Maximum HQs for Dietary COPCs based on individual prey species.

Section 8.1.2, COPCs Evaluated: Screening tables for the SLERA and refined screen should be provided so it is completely transparent how the receptor / COPC pairs were identified for birds and mammals. It is not clear how the list in Table 8-1 was developed.

Section 8.1.3., Table 8-8, Predicted Prey Tissue Concentrations and Attachment 4, Table 7-2: The use of the mechanistic model, which predicts average concentrations site wide, underestimates concentration for localized beaches smaller than the site. In addition, the text and attachment are not clear about what sediment concentration was used to predict "average prey concentrations". This needs clarification. Instead, BSARs or BSAFs should have been used to predict tissue concentrations of sediment invertebrates like clams and worms using sediment form localized beaches at the site identified in Table 8-7, which was been done in previous versions of the risk assessment.

Section 8.1.4.1 and Table 8-10, Selected Dietary TRVs and Table 8-13: In addition to high molecular weight PAHs (HPAHs), a TRV should have been developed and applied for low molecular weight PAHs (LPAHs) from the same source (EPA Eco SSLs). The NOAEL TRV is 65.6 mg/kg bw/day) - a LOAEL can be derived from the database just as was done for the other COPCs (e.g. HPAHs). LPAHs were detected in fish and invertebrate tissue.

Section 8.1.5.1, Spotted Sandpiper and Map 8-1: A 2-mile beach exposure area is outlined in Table 8-16. However, sandpiper would be using a linear shoreline and crossing the river would be unlikely. Therefore, if a larger area is considered, it should be ***2 miles on one side of the river***, which is also consistent with contiguous sources. Table 8-16 indicates river mile stretches on both sides as well as Map 8-1. This methodology diluted and bi-furcated shorebird habitat as well as sources and should be revised appropriately.

Section 8.1.5.1, Table 8-16, Total TEQ: Total TEQ HQ should be the addition of dioxin /furan TEQ and PCB TEQ. However, for beach area B14-B24 the two values do not add up. PCB TEQ HQ is reported as 11 and dioxin /furan TEQ reported as 17, which should equal 28 and not 20.

Section 8.1.5.1.1, Spotted Sandpiper, Use of predicted rather than measured concentrations in prey species: The text states "the absence of a relationship between sediment and tissue concentrations

means that there is not a relationship between dietary risk (should it occur) and sediment concentrations". This is not accurate – it only means a relationship could not be developed between sediment and tissue not dietary risk. This should be highlighted as a data gap where prey tissue should be collected directly from these areas to refine risk estimates.

Section 8.1.5.1.2, Hooded Merganser: COPCs with HQ>1 based on step 2 (EPA problem formulation) should be identified in table format along with locations, as is done in Table 8.17 for multiple prey portions (step 3). Information from Attachment 17, Table 3-1 and 3-2 should be brought to the main text and it should be clear what additional areas are identified.

Section 8.1.5.1.2, Bald Eagle: COPCs with HQ>1 based on step 2 (EPA problem formulation) should be identified in table format along with locations, as is done in Table 8.18 for multiple prey portions (step 3). Information from Attachment 17, Table 4-1 and 4-2 should be brought to the main text and it should be clear what additional areas are identified. In addition, ***bass should have been used as a dietary item for the bald eagle, since it is the only fish receptor collected on a 1 mile exposure basis and per EPA's Problem Formulation***. Or, look to the osprey diet, which did include smallmouth bass, for this information???

Section 8.1.5.1.3, Bald Eagle, Mercury: This section dismissed mercury as a basin wide contaminant. However, the text should be transparent that there are Site sources of mercury, namely Willamette Cove, where the highest mercury was detected in northern pikeminnow tissue. Mercury comparisons in Figure 8-2 are biased by the larger fish (e.g. smallmouth bass) collected upstream.

Section 8.1.5.1.4, Osprey: COPCs with HQ>1 based on step 2 (EPA problem formulation) should be identified in table format along with locations, as is done in Table 8.17 for multiple prey portions (step 3). Information from Attachment 17, Table 5-1 and 5-2 should be brought to the main text and it should be clear what additional areas are identified. ***Also, carp, largescale sucker, pikeminnow and brown bullhead were collected and composited over 3 mile stretches of the river for all wildlife species. However, when these data were used in the osprey (and eagle, mink, etc) assessment (Attach 17, Table 5-1), the 3 mile composites were averaged site wide according to footnote "b" of Table 5-2. This should be revised to be in 3 mile segments.***

Section 8.1.5.1.5, Mink, Lead and Antimony: It is not defensible to attribute risk to antimony and lead to a fish sinker in a composite without re-collecting the sample. Also, sources in the area such as Gunderson could contribute to this result.

Section 8.1.5.1.5, Mink COPCs: COPCs with HQ>1 based on step 2 (EPA problem formulation) should be identified in table format along with locations, as is done in Table 8.17 for multiple prey portions (step 3). Information from Attachment 17, Table 6-1 and 6-2 should be brought to the main text and it should be clear what additional areas are identified.

Section 8.1.4.1.6, River Otter: COPCs with HQ>1 based on step 2 (EPA problem formulation) should be identified in table format along with locations, as is done in Table 8.17 for multiple prey portions (step 3). Information from Attachment 17, Table 7-1 and 7-2 should be brought to the main text and it should be clear what additional areas are identified.

Section 8.1.5.2.1, Evaluation of Varying Prey Portions: Additional analysis was provided here beyond the previous version of the ecological risk assessment. The relative proportion of prey in the diet of wildlife

receptors was evaluated using probabilistic risk assessment methods. If probabilistic methods were to be used in the BERA, the methods were to be approved by EPA first. It is unclear why the osprey was exclude from this analysis. However, the dietary prey fraction ranges considered were too narrow to provide useful information (see Table 8-25), and results in the sensitivity analysis ***only identifying potential exposure concentrations ranges lower than already assumed using deterministic methods (for all but bald eagle) and not representative of the entire potential range***. However, it is interesting to note that even using these narrow ranges for evaluating sensitivity it appears as if the ***bald eagle and mercury exposure risk estimates are not protective of likely exposure.*** These should be updated in the risk assessment. If the probabilistic methods are included, this should be re-done with coordination with EPA.

Section 8.1.5.2.2, Belted Kingfisher: COPCs with HQ>1 based on step 2 (EPA problem formulation) should be identified in table format along with locations, as is done in Table 8.17 for multiple prey portions (step 3). Information from Attachment 17, Table 8-1 and 8-2 should be brought to the main text and it should be clear what additional areas are identified.

Section 9.0, Amphibian Risk Assessment: It is unclear why the dioxin / furan water quality criteria was removed from this assessment. This should be used to assess water concentrations of dioxins and furans to amphibians and other aquatic life.

Section 9.1.3.4, Surface Water EPCs: Peristalic pump samples should be added back into the exposure dataset. Also, site wide exposure point concentrations are of limited use for assessing amphibians which are exposed and reproduce over smaller spatial scales than the entire site.

Section 9.1.4.1, Risk Characterization Results and Uncertainty: AWQCs should be used in addition to the alternative water TRVs. Instead, the risk characterization is only based on the alternative water TRVs.

STOPPED HERE

FPM Questions:

1. Chemical List for FPM: What are the data rules for the development of the chemical list in this version of the risk assessment? The chemical list is perhaps the most critical piece of the use of the floating point model. Since the chemical list affects the output and values of the FMP model, disagreement at this stage represent a fatal flaw in the FPM. At this point, there are several discrepancies in the decision framework used to develop the list.

    a. It appears that several chemicals showed significant difference between hit and not hit distributions were not included because according to the text they did not exceed the SQG used in the SLERA. This has not been a decision rule used in previous versions of the predictive models. Any chemical detected 30 times or more that shows statistical difference between the hit and not hit distributions should be included. The goal of the

predictive models is to develop ***site specific models*** that correlate with toxicity and not pre-determine the chemical list using non-specific criteria.

i. Antimony
ii. Dibutylphthalate
iii. Diethylphthalate
iv. Dimethylphthalate

b. It appears some chemicals without SQGs were not included in the floating point model. This is not consistent with objectives to determine a site specific model and is not consistent with previous versions of the model.
   i. Butyltin
   ii. Tetrabutyltin

c. It appears that chemicals were added to the model that did not appear to have any correlation with toxicity according to the analysis between hit and no hit distributions provided. On what basis were these chemicals included? Chemicals not associated with toxicity can affect the SQVs for other chemicals in an inappropriate way. This is especially important if they co-vary such as Endrin and DDX compounds.
   i. Endrin
   ii. Endrin Ketone

d. It appears that chemicals initially included in the model were removed because they were not correlated with toxicity. If the hit and no hit distributions are statistically different then the chemical is correlated with toxicity. Please clarify the decision framework to remove lead. Previous versions have used measures of reliability as justification. However, overall error and reliability rates are not the only measure of interest. Furthermore, if a chemical is removed from as a relevant SQG, it must be removed and the model re-run without that chemical. Otherwise, the inclusion influences the SQVs for other chemicals in the model inappropriately. In addition to the concern above, the removal of lead this appears to have been made analyzing Level III threshold effect reliability and not Level II. Please explain.
   i. Lead (included in all previous versions of the model)

e. Chemicals determined to be non-CERCLA chemicals in the document were not considered in the predictive models. This is not appropriate as these chemicals have been found by both LWG and Government Team models (NOAA) to be highly correlated with toxicity.
   i. Diesel-Range Hydrocarbons
   ii. Residual-Range Hydrocarbons

f. Conventional: Previous versions of the model have included %fines and organic carbon. This version includes only ammonia and sulfides. Presentation of significance between hit and no hit distributions are not included for additional conventional such as fines and OC and it is unclear why these were not included in this version of the model. Ammonia and sulfides are highly associated with contaminated areas and likely co-vary significantly with other contaminants in the model. Why were these included?

2. Statistical Difference Between Hit and No Hit Distributions: The use of parametric methods (ANOVA) have been shown to be inaccurate in determinations of significant difference between

distributions. DEQ's review has bound that these distributions are often non-normal and the variances are not equal. In the statistical tests between hit and no hit distributions it appears the data were log transformed for the non-parametric tests. This doesn't appear to be necessary and appears to negate the advantages of using a non-parametric model. Can you explain why this was done?

3. Chemical List by Species and Endpoint: The determination of statistical significance and associated chemical list should be species and test specific. Instead, the chemical list is the same between endpoints and species when tests between hit and no hit distributions between the two show differences. A separate chemical list should be developed based on statistical difference of hit / no hit distributions for each endpoint. Why was this not completed?

    a. There are Excel spreadsheets attached to the BERA that includes analysis of significant difference between the hit and no hit distributions. Some of the headers indicate HG or Hyella growth. It is unclear if this indicates biomass in all cases. Please clarify.

4. Predictive Models and Risk versus Risk Management: There is text in the document that indicates the "balanced model" (balanced in terms of false negatives and positives), is also the model used to predict risk. This is not correct and should be clarified in the document. However, predictions should be made on the full range of risk as defined and presented in EPA's problem formulation. This includes Levels 1,2,and 3 thresholds to lower false negative rates than is achieved by using the balanced model. Where are the floating point model runs and results for predicative models other than the balanced model?

5. Comprehensive Benthic Approach: Where are the details presented on this management approach? Is this presented in another document such as the FS or additional management document?

6. Evaluation of Reliability for FPM Model SQVs: An evaluation of reliability presented individually based only for each endpoint and species separately is misleading. Although SQVs can be ***developed*** for each endpoint separately, the evaluation of ***reliability*** should include the combination of these 4 sets of SQVs into one SQV.

7. Mean Quotients: The text accurately states "once that set is determined, the SQVs must be used together to predict the toxicity of the contaminant mixture – they are not independent. Each SQV explains toxicity along with all the other SQVs that were derived from the model…". While we are in agreement with these statements, it is unclear why specific individual FPM SQVs were used outside the context of the rest of the model set in mean quotient analysis? Please explain.

Attachment 4, Table 7-2: It is unclear why only the average concentrations in shorebird prey was predicted. It appears the average sediment concentration was used to calculate an average worm and clam prey concentration for shorebirds, which is not appropriate. Each shorebird area should be calculated separately and encompassing the range in concentration as indicated by the range presented in this table.